Exp Brain Res (2014) 232:1233–1240
DOI 10.1007/s00221-014-3839-7

RESEARCH ARTICLE

Exploring the world with Bálint syndrome: biased bottom-up guidance of gaze by local saliency differences

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Received: 13 November 2013 / Accepted: 9 January 2014 / Published online: 22 January 2014

Abstract Bálint syndrome is a combination of severe deficits affecting spatial attention, visuo-motor control and oculomotor function. While the severe restriction of attention (simultanagnosia) and impairments of visually guided reaching have been extensively studied, oculomotor apraxia has received comparatively little attention. The main explanatory hypothesis of oculomotor apraxia is that it is a direct consequence of the severe restriction of attention. Here, we examined in a patient with Bálint syndrome to what extent local image features such as luminance and contrast predict whether a region will be fixated or not. During the viewing of natural photographs, the patient made saccades of very small amplitude, but showed strongly increased fixation duration. In addition, the horizontal and vertical range of fixations was severely restrained compared to control subjects. When analysing the local feature content at fixation, we found that central fixations of the patient contained less local luminance and contrast than fixations of controls while he made fixations to peripheral image regions with disproportionately high luminance and contrast. These findings suggest that while our patient gazes at central regions irrespective of their local feature content, he only looks to the periphery when his gaze is captured by particularly conspicuous features. We propose that oculomotor apraxia in Bálint syndrome reflects a combination of biased representations within a parietal priority map and increased fixational activity due to biased interactions within the oculomotor network.

Keywords Bálint syndrome · Eye movements · Parietal lobe · Saccade planning · Simultanagnosia · Spatial attention

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Introduction

Bálint syndrome, as originally described by Bálint (1909; traduction in Harvey 1995), is characterized by a combination of visual–spatial disturbances including severe constriction of attention, often around a single object (simultanagnosia, which Bálint termed 'psychic paralysis of gaze'), errors in visually guided pointing or reaching (optic ataxia) and a spatial disorder of attention. Following the initial description, many authors also noted impaired initiation and control of saccadic eye movements in patients with Bálint syndrome (oculomotor apraxia; Luria et al. 1963; Girotti et al. 1982; Rafal 1997; Rizzo and Vecera 2002). Oculomotor apraxia is evidenced as a failure to disengage gaze from a fixated object (sometimes described as 'sticky' fixation), making it often impossible to look at objects shown in the visual periphery, or conversely the failure to maintain fixation on a given object. While numerous experimental studies explored the cognitive bases of simultanagnosia and optic ataxia, oculomotor disturbances of Bálint patients have comparatively rarely been studied. The obvious reason is that the calibration of eye-tracking equipment requires stable fixation of sequentially presented targets, a capacity lacking in most patients with oculomotor apraxia

(Rizzo and Hurtig 1987; Ptak and Müri 2013). Therefore, only few patients have been examined using eye tracking, and these probably represent a selection of relatively less impaired cases. Some of these patients are unable to follow a slowly moving stimulus (Girotti et al. 1982) and show a seemingly chaotic fixation pattern when asked to freely explore natural stimuli (Luria et al. 1963). More recent studies found that simultanagnosic patients fail to fixate informative regions (such as the clock hands when reading a clock, or the eye regions when gazing at faces; Nyffeler et al. 2005; Dalrymple et al. 2011), suggesting that irrelevant perceptual details capture the gaze of these patients and thus lead to an erratic fixation pattern. Based on this observation, some authors have hypothesized that local saliency differences inherent in natural images capture the gaze of simultanagnosic patients and thus determine the observed, chaotic fixation pattern (Nyffeler et al. 2005). On the other hand, top-down control over eye movements may partly be preserved, and at least some patients are able to fixate on specific image regions when instructed to do so, even though they fail to do it spontaneously (Dalrymple et al. 2013b; Jackson et al. 2009). This finding suggests that during spontaneous scene viewing bottom-up factors dominate and are the main determinants of whether an image region is fixated or not. However, so far no study examined the local content of image regions fixated by patients with Bálint syndrome.

Here, we measured ocular fixations of a Bálint patient asked to freely explore photographs of natural scenes and then examined the local statistics of fixated image regions. Previous studies with healthy participants using this approach identified local contrast (Reinagel and Zador 1999; Parkhurst and Niebur 2003) and edge density (Mannan et al. 1996; Tatler et al. 2005) as reliable predictors of whether a region was fixated or not. We therefore focused our analysis on these two features and additionally examined whether local luminance and colour differences were particularly high or low at fixated regions. Our findings show that local image features affect ocular fixations in Bálint syndrome qualitatively differently compared to healthy participants and thus suggest biased bottom-up processing during ocular scanning.

Methods

Patient description

ER, a former cook without special education, suffered from multiple strokes at the age of 66 when undergoing coronary surgery. Upon awakening from anaesthesia, he showed slight left hemiparesis and complete right hemianopia, as well as severe visual–spatial disturbances described in detail below. Structural MRI revealed ischemic lesions of the right dorsal frontoparietal cortex, the left medial occipital cortex including primary visual cortex and the lateral occipitotemporal cortex on both sides (Fig. 1a). Medial occipital damage to the left hemisphere extended from the calcarine sulcus dorsally into the precuneus and to the posterior intraparietal sulcus. Right frontoparietal damage

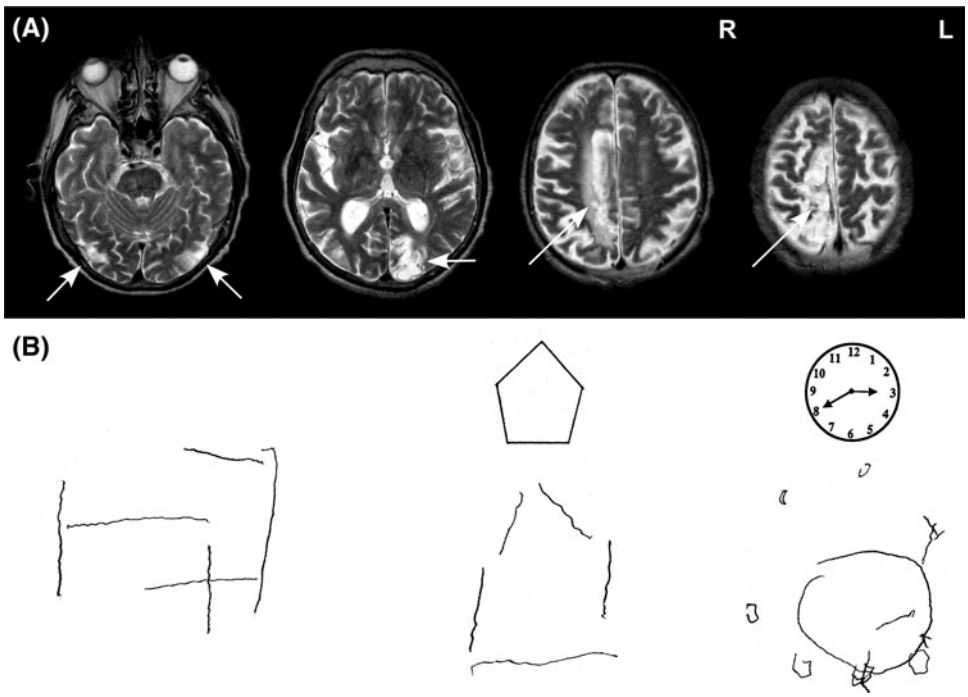

Fig. 1 **a** T2-weighted MRI scan performed 9 months post-injury showing ischemic damage to bilateral occipito-temporal cortex (*left*), left medial occipito-parietal cortex (*middle*) and dorsal frontoparietal cortex (*right*). **b** ER's attempts to draw a cube from memory (*left*) and to copy a pentagon and a clock face

extended posteriorly into the superior parietal lobule and the anterior intraparietal sulcus.

ER initially exhibited all signs of Bálint syndrome including optic ataxia, simultanagnosia, oculomotor disturbances and severe visual–spatial confusion, making it difficult for him to identify visually presented objects. The present examination was performed 7 weeks following the stroke when ER was able to fixate an object for several seconds. During this period, ER benefited from 1 to 2 daily sessions of therapy targeting compensation of oculomotor deficits (ocular pursuit tasks, visual search and detection of visual targets presented in the left or right visual field) and optic ataxia (pointing to dots presented on a touch screen, reaching and manipulating different objects). He was fully oriented and showed no signs of aphasia or apraxia. Out of 35 common household objects presented as line drawings, he named 28 correctly and made visual errors for the remaining objects (e.g. calling a screw a 'little key'). He scored within average range on verbal fluency, verbal abstraction (similarities subtest of WAIS-III) and oral arithmetic, though memory was moderately impaired (six of ten words recalled after 30 min). Confrontation testing showed complete right hemianopia (formal perimetry testing was impossible because ER was unable to maintain fixation during the examination). Optic ataxia was tested clinically by asking ER to point to the examiner's finger and to grasp a pencil held out by the examiner in his visual periphery (five trials per hemifield) while maintaining fixation on the examiner's nose. ER showed deviations of approximately 2–5 cm when pointing in his left hemifield, but much larger errors (10–15 cm) in his right hemifield. Optic ataxia was also manifested in ER's drawings, where he was often unable to position the pencil correctly (e.g. when asked to cross out visual targets). The patient additionally showed moderate oculomotor apraxia characterized by 'sticky' fixation (often fixating a face or an object for several seconds) and impaired ocular pursuit (loosing contact with the visual target, in particular for movements to the right). Simultanagnosia was evidenced when ER was asked to count dots displayed on a sheet of paper or to name several objects arranged on the table. In the latter situation, he would comment that the objects became all intermixed and that it was impossible for him to disentangle the individual items. He was able to name individual letters, but failed to read even short words (e.g. he identified the French word 'LUNE'—moon as 'LURO', and the word 'VIOLONISTE' as 'VOLVOLINE'). Copying simple shapes or drawing them from memory was extremely difficult for ER as he was unable to position single elements of the drawing correctly and quickly lost track once he lifted the pencil (Fig. 1b). Table 1 shows ER's performance in tests probing visual, visual–spatial and constructional abilities. He was impaired in all tests except those that required single-object identification or matching. At the time of this examination, ER did not show signs of left spatial neglect, but missed many cancellation items in both hemifields. In other tasks or during therapy, he would generally detect target objects that were to his left better than those located on the right. He also did not show clear head or gaze deviation to the right.

Table 1 ER's scores in visual and visual–spatial tests

Test battery	Subtest	Score	Below cut-off/5 %
VOSP	Screening	20	
	Incomplete letters	9	X
	Silhouettes	14	X
	Object decision	11	X
	Progressive silhouettes	14	
	Dot counting	4	X
	Position discrimination	10	X
	Number location	1	X
	Cube analysis	1	X
WAIS-III	Block design	0	X
BORB	Length match	19	X
	Size match	24	X
	Orientation match	19	X
	Position of gap match	26	X
	Minimal feature match	21	
	Foreshortened match	20	
	Object decision	19	X
BIT	Albert test, omissions left (of 18)	9	X
	Albert test, omissions right (of 18)	8	X
	Star cancellation, omissions left (of 27)	19	X
	Star cancellation, omissions right (of 27)	15	X

BIT behavioural inattention test (Wilson et al. 1987), *BORB* Birmingham object recognition battery (Riddoch and Humphreys 1993), *VOSP* visual object and space perception battery (Warrington and James 1991), *WAIS-III* Wechsler Adult Intelligence Scale (Wechsler 1997)

Despite his severely impaired exploration of large, cluttered visual displays (such as typically present in cancellation tasks), ER accurately detected items defined by colour or shape when they were presented in his central visual field. We tested him using a simple visual search task containing four squares that were either red or green and filled or unfilled. The task was to indicate whether the display contained a square defined by the combination of colour/filledness (e.g. a green/unfilled square) while the three distracters were systematically varied. In the *dissimilar* condition, all distracters differed on both features from the target (e.g. all three were red/filled). In

the *similar* condition, all distracters shared one feature with the target (e.g. all three were green/filled). In the *mixed* condition, one distracter shared one feature with the target (e.g. green/filled) while the other two distracters shared the other feature (e.g. red/unfilled). Figure 2 shows the results of ER compared to a group of ten age-matched controls and 14 patients with left neglect tested in a previous study (Ptak and Valenza 2005). ER made no omission on this task and only one false response when the target was absent. Both control groups made more omissions (controls: 2.7 %, neglect: 5.1 %) and false responses (controls: 4.6 %, neglect: 1.5 %). ER's reaction time data were analysed with an ANOVA with factors visual field (left/right) and condition (mixed, similar, dissimilar). The analysis revealed only a significant main effect of visual field, with slower reactions to targets shown in the right (hemianopic) compared to the left hemifield [$F(1,137) = 6.03$, $p < .05$]. We compared ER's data directly to controls and neglect patients using a Bayesian approach (Crawford and Garthwaite 2007). Though ER was overall slower than healthy controls, none of the comparisons reached significance. In contrast, he was significantly faster than neglect patients to detect left hemifield targets in the mixed condition ($p < .05$). This finding shows that ER processed visual targets presented in his central visual field adequately and with normal speed. Any differences between him and control participants in visual exploration of natural images can therefore not be attributed to impaired attention for information presented at fixation.

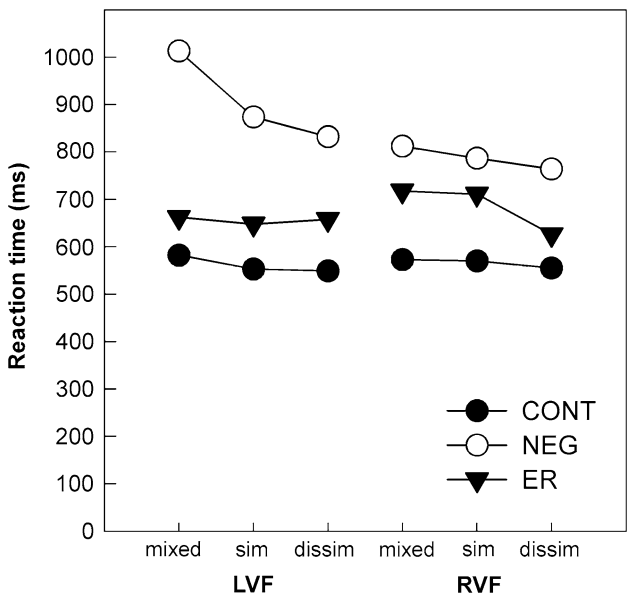

Fig. 2 Results of the visual search task of ER compared to healthy controls and neglect patients (*LVF*/*RVF* left/right visual field)

Material and procedure

All participants gave written informed consent and the study was approved by the Ethical committee of the University Hospital, Geneva. Material and procedure were as described in a previous study (Ptak et al. 2009), which involved 18 healthy controls and 13 right-hemisphere stroke patients (six without and seven with left spatial neglect). Twenty colour photographs depicting roughly symmetrical portraits of natural scenes, architecture or regular patterns were shown on a 21″ CRT for 15 s each. The fixation position of the right eye was measured with an infrared, video-based eye-tracker (HighSpeed; SMI, Berlin, Germany) at a sampling rate of 240 Hz. During calibration, ER was required to fixate sequentially on nine small circles presented at different positions. The calibration procedure was under manual control of the experimenter who indicated verbally the position of the current target and verified visually whether eye position was stable. If necessary, calibration was repeated for specific calibration targets, and a verification run was performed to ensure that calibration was adequate. In order to favour bottom-up guidance of gaze, no specific instructions were given to ER other than to freely explore each image.

Analysis

Saccades and fixation locations were extracted offline using velocity (saccade: ≥50°/sec) and duration (fixation: ≥100 ms) criteria. Spatial and temporal parameters of saccades and fixations were then computed for each participant. Local image features were extracted from patches of 1° × 1° drawn around each fixation and computed as described previously (Ptak et al. 2009). Briefly, local luminance was calculated as the average intensity of all pixels within the patch scaled to the mean luminance of the whole image. Following scaling, values >1 indicated that the patch was brighter than and values <1 that it was relatively darker than the average brightness of the image. Chromatic contrast was expressed as the standard deviation of pixel intensities in each RGB colour channel, which were then averaged and finally normalized to the maximal possible contrast. Luminance contrast was computed similarly to chromatic contrast, but only with one channel (grey). Finally, edge content was extracted by convolving the original image using the 'canny' edge detection algorithm (Canny 1986) implemented in Matlab® Image Processing Toolbox.

Results

Table 2 shows the results of basic saccade and fixation parameters of ER as compared to 18 healthy participants

Table 2 Means (±SD) of basic saccade parameters

Group	Saccades, total (*N*)	Saccades, leftward (*N*)	Saccades, rightward (*N*)	Saccade amp (°)	Fixation time (ms)
Controls	35.6 ± 6.4	15.6 ± 2.8	15.3 ± 3.3	4.8 ± 0.8	240 ± 42
Neglect	32.1 ± 6.8	12.6 ± 3.6	14.3 ± 3.5	3.3 ± 0.9	231 ± 61
ER	16.1 ± 5.2	7.8 ± 3.5	6.3 ± 2.3	2.8 ± 0.8	547 ± 186

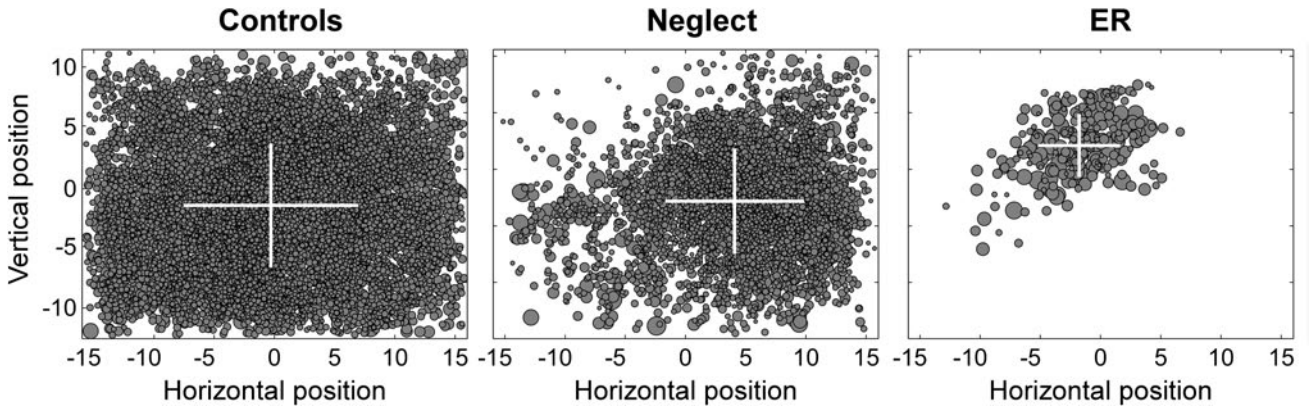

Fig. 3 *Scatter plot* showing all fixations produced by ER as compared to healthy controls and neglect patients. The size of circles representing each fixation is proportional to fixation duration. The *white cross* indicates the mean ± 1 SD of the horizontal and vertical distribution of fixations

and seven neglect patients tested in our previous study (Ptak et al. 2009). Statistical comparisons were performed using a Bayesian approach for small samples. The aim of this approach is to reduce the probability that the patient score is falsely classified as being abnormal (Crawford and Garthwaite 2007). It provides a point estimate of the abnormality of the patient's score relative to a control population by treating population parameters (such as the mean) as random variables with a probabilistic distribution. In comparison with the control ($p < .01$) and the neglect group ($p < .05$), ER produced a lower total number of saccades and less leftward (controls: $p < .05$; neglect: ns.) or rightward (both $p < .05$) saccades. He had a slight directional bias, making significantly more saccades to the left than saccades to the right [$t(14) = 2.3$, $p < .05$]. His mean saccade amplitude was significantly smaller than controls ($p < .05$), but did not differ from neglect patients. Finally, he exhibited significantly longer fixation durations than the control and the neglect group (both $p < .01$).

Figure 3 shows a scatter plot of all fixations made by ER as compared to controls and neglect patients. The average fixation position was nearly central in healthy controls (horizontal: -0.3 degrees; vertical: −1.2°), shifted to the right in neglect patients (horizontal: 4.4°; vertical: −0.7°) and shifted upwards and sligthly to the left in ER (horizontal: −1.9°; vertical: 4.5°). As can be seen in Fig. 3, the dispersion of fixations was much smaller in ER compared to both groups. Most of his fixations were restricted to a sector that covered −7 to +5 degrees horizontally and 0–7° vertically. In contrast, healthy controls had a range in the horizontal and vertical direction that covered the entire image surface, and neglect patients also exhibited a larger range than ER—even in the horizontal direction and although their average horizontal position was shifted to the right.

In order to examine whether local image content affected the distribution of ER's fixations, we extracted statistics for luminance, contrast, chromatic contrast and edge content from image patches drawn around each fixation. Previous work has shown that the distribution of local features in photographs tends to increase or decrease towards the edges (Parkhurst and Niebur 2003). We therefore computed quadratic polynomials for each of the four image features across horizontal fixation positions. Figure 4 shows polynomial functions of ER as compared to healthy participants and neglect patients. As reported previously (Ptak et al. 2009), neglect patients tended to look at regions located in the left half of the image only when these regions had particularly high luminance, but low edge content. The polynomial functions generated from ER's data were strongly U-shaped and showed significant deviations ($p < .01$) from the pattern of control participants for all four local features. For local luminance and luminance contrast, predicted feature values were *below* control values for central positions, but significantly *above* control values for more

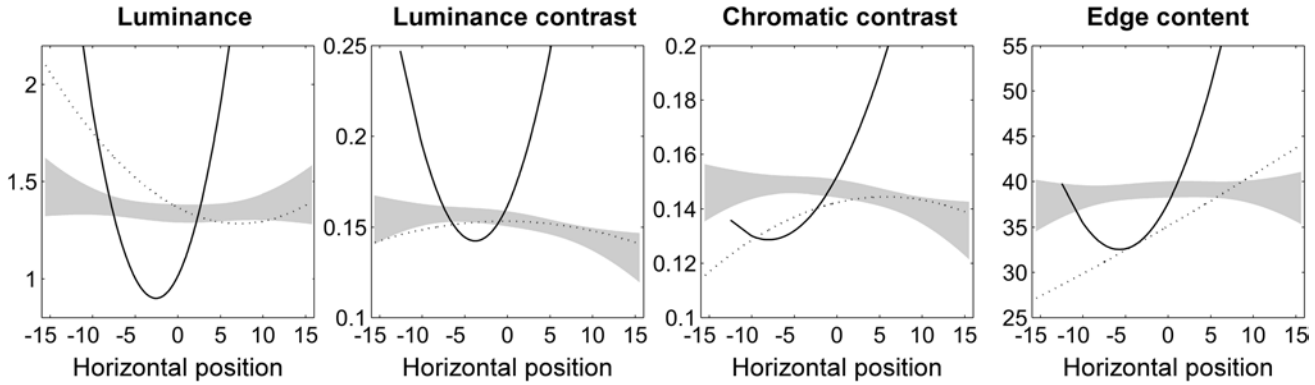

Fig. 4 Polynomial functions predicting local feature content across horizontal positions of fixations. The *grey* area is based on the 99 % confidence interval of healthy controls. *Stippled line* neglect patients; *full line* patient ER

peripheral positions. The trend was similar for chromatic contrast and edge content, with the difference that only right peripheral fixations were directed to regions with particularly increased local feature values. Thus, ER gazed at central regions irrespective of their local feature content, but only looked to the periphery at regions characterized by particularly conspicuous features. In addition, we examined whether local feature content affected ER's first fixation similarly to control participants. For this analysis, we compared ER to healthy controls, neglect patients and a random observer by treating ER's first fixations for all images as if they represented a group of independent observations. Analysis of variance revealed a significant difference between groups for local luminance [$F(3,54) = 3.49$, $p < .05$], indicating that healthy controls and neglect patients fixated regions of higher luminance compared to random (LSD-tests, $p < .05$). In contrast, the luminance of ER's first fixations did not differ from a random observer. No significant differences between control groups, ER and the random observer were observed for luminance contrast [$F(3,54) = 1.39$], chromatic contrast [$F(3,54) = 1.77$] and edge content [$F(3,54) = 2.09$].

Discussion

We identified several features of oculomotor apraxia in a patient with Bálint syndrome that extend previous observations. ER's pattern of basic saccade and fixation parameters strongly differed from healthy controls and also differentiated him from neglect patients. He made significantly less saccades than both groups and had strongly reduced saccade amplitude than healthy controls. However, the latter observation does not appear to be limited to Bálint syndrome as neglect patients exhibit a similarly reduced saccade amplitude during visual exploration. The most significant difference to controls and neglect patients, and one that is directly related to the notion of 'sticky' fixation in Bálint syndrome, is ER's significantly prolonged fixation duration. On average, ER produced fixation durations that were more than the double of those of control participants. Similar durations were observed in a previous study when a simultanagnosic patient was required to make saccades to peripheral targets while a central fixation stimulus was present (a so-called overlap task; Nyffeler et al. 2005). However, the sudden appearance of a stimulus at fixation also strongly affects fixation durations in patients with unilateral posterior brain damage and spatial neglect (Ptak et al. 2007; Walker and Findlay 1996), while the prolonged durations during visual scanning appear to be specific to Bálint syndrome.

How can 'sticky' fixation during ocular exploration be explained? Some authors (Rafal 1997; Rizzo and Vecera 2002) proposed that oculomotor apraxia is due to a pathological constriction of visual attention to a single object. According to this hypothesis, patients make erratic eye movements because of a reduced 'spatial window' of attention (Dalrymple et al. 2013a) and consequently the failure to disengage attention from a fixated object. Given the close interdependence of attention and saccade programming (Remington 1980; Hoffman and Subramaniam 1995), a reduced 'spatial window' of attention would strongly affect the selection of saccade targets. Our analysis of local image content at fixated locations only partly supports this conclusion. ER only made saccades to peripheral locations when these had high luminosity and contrast. Given that ER had a right homonymous hemianopia, it is important to determine to what extent this finding can be explained by his visual field impairment, since for all eye movements directed to the right, the saccade landing position was not visible for him. We would therefore expect that fixations located on the right side be selected randomly, which predicts a strongly asymmetrical distribution of local image features between left and right fixations. Contrary to this

prediction, the pattern of increased local luminosity and contrast was very similar for left and right peripheral locations and is therefore not adequately explained by ER's right hemianopia. Rather, it suggests that ER's gaze is only captured by image regions that are particularly conspicuous. Although factors such as task constraints and expectation significantly influence visual exploration during active scene viewing (Tatler et al. 2011), bottom-up visual saliency is a powerful predictor of fixation locations (Itti and Koch 2000; Itti et al. 1998; Parkhurst et al. 2002). Our findings suggest that in Bálint syndrome, this role of local saliency differences is biased: for central positions, ER-fixated image regions that were relatively less conspicuous compared to healthy participants. Less conspicuous central regions should make it easier to shift attention (and gaze) away to the periphery, yet ER only looked at peripheral locations when these were disproportionately salient. In our view, this finding reflects two possibly interacting factors. On the one hand, biased attentional priority following damage to posterior parietal cortex (PPC) along the intraparietal sulcus (IPS). Neurophysiological studies have shown that the IPS encodes stimuli in a feature-independent manner and integrates bottom-up saliency signals with top-down task-related factors into a spatiotopic priority map of the environment (Bisley and Goldberg 2010; Gottlieb et al. 1998; Ptak 2012; Vandenberghe et al. 2012). Attentional priority may be conceived as emergent property computed from converging inputs from different sensory modalities and relevance signals originating in prefrontal cortex (Ptak and Fellrath 2013). If the priority map is crucial for the selection of sensory contents by attention, damage to this representation, in particular if it is bilateral, should have devastating consequences on spatial attention. This is indeed what happens in patients with Bálint syndrome, who in severe cases are described as virtually blind due to their failure to select stimuli for conscious processing (Rizzo and Vecera 2002; Holmes and Horrax 1919; Kim and Robertson 2001). However, it is unclear why bilateral damage to the parietal priority map should result in a strong ocular bias towards central regions (or 'sticky' fixation). This central bias is more readily explained by functional impairment of structures of the oculomotor network that are involved in fixational activity. The mesencephalic superior colliculus contains neurons that discharge when a stimulus is actively fixated, while other neurons become active when a saccade is prepared and executed (Munoz and Wurtz 1993; Dorris et al. 1997). This structure is directly connected to the PPC, and unilateral parietal damage has facilitatory effects on the ipsilateral and inhibitory effects on the contralateral colliculus (Sprague 1966; Rafal 2006). Following bilateral damage to the PPC top-down facilitatory influences of the parietal cortex on the superior colliculus is diminished, making it difficult to initiate saccadic eye movements; as a consequence, fixational activity in both colliculi is disinhibited and leads to the strong bias favouring stimuli presented at fixation. Such a mechanism could therefore underlie the 'sticky fixation' observed in patients with oculomotor apraxia. However, this model, though supported by neurophysiology and some experimental studies on animal models of spatial neglect (Payne et al. 1996; Rushmore and Payne 2003), awaits direct support by human lesion studies.

In sum, based on our findings, we propose that oculomotor apraxia, in particular the bias of Bálint patients towards stimuli shown at fixation, reflects a combination of a severe impairment of mechanisms involved in attentional selection and a low-level oculomotor impairment following biased interactions between the PPC and the superior colliculus.

Acknowledgments Study supported by the Swiss National Science Foundation (Grant 320030-134591) and the De Reuter foundation (Geneva).

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